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**Tumors of Lung.**—Tumors involving the apex of the lung may cause considerable pain in the shoulder. In fact this pain may be the only symptom of which the patient complains. Three cases are described in which the shoulder pain was accompanied by definite roentgenological evidence of supraspinal bursitis and it was not until after the usual therapeutic methods for this disease had failed that a tumor involving the apex of the lung was discovered. It should be routine to examine the chest of all patients with shoulder pain even though an apparently sufficient local lesion has been found. The 3 reported cases showed more or less extensive calcification about the shoulder with associated neoplasm of the lung.—*Pulmonary Apical Tumefaction Simulating Bursitis. Necessity for Routine Chest Examination in Patients with Shoulder Pain, L. Nathanson, Quart. Bull. Sea View Hosp., April, 1939, iv, 326.*—(*A. A. E.*)

**Aspiration Biopsy of Lung Tumors.**—By aspiration biopsy is meant the insertion of an aspirating needle directly into the tumor in order to obtain a plug of tissue for smear and quick stain. Histological evidence of cancer is of importance and a decided advantage in the proper management of a patient with a primary tumor of the lung. One is not justified in aspirating a lesion, suspected of being a metastasis, until an exhaustive search has failed to locate a primary cancer elsewhere in the body. Aspiration biopsy is particularly valuable for tumor far out in the parenchyma beyond a primary or secondary bronchus and usually inaccessible for bronchoscopic biopsy. Aspiration is used in those cases where roentgenographic evidence indicates that the tumor is not accessible by bronchoscopy or where the condition of the patient indicates an unfavorable reaction to bronchoscopy. The exact site of puncture can be outlined on the skin under roentgenoscopy, as well as size and depth of the tumor and angle of inclination of the needle. Needles of 17 or 18 gauge calibre and measuring 10, 15, and 20 cm. in length, should be available. A sense of touch is soon developed that can distinguish between lung tissue and the "leathery feel" of tumor tissue. Constant suction is applied after the needle enters the tumor. The needle is inserted a centimetre or more and then rotated to loosen tissue. Particles of suspected tumor tissue are smeared between slides and blood that may be aspirated is allowed to clot and saved for sectioning. Patients in this series exhibited no infections, pleural shock, or mediastimal shifting, unusual pain, cough or haemorrhage. Pain, cough, haemoptysis and partial collapse of the lung occasionally occurred, and were readily controlled. There was one case of probable air embolism. Aspiration biopsy on 92 different pulmonary lesions produced material sufficient to establish a positive diagnosis of carcinoma in 56 or 60.8 per cent of the 67 cases with positive aspirations, 56 were carcinoma, 4 were malignant tumors and 7 were benign lesions. There were 6 proved failures in the series. Two of these were

later diagnosed as carcinoma by bronchoscopic biopsy, 2 by biopsy of lymph nodes and 2 by autopsy. Thirty-two cases of primary carcinoma were proved by aspiration biopsy following one or more bronchoscopic failures. Of 130 cases of clinical primary carcinoma of the lung, between 1927 and 1937, biopsied by all methods, 56 were proved by aspiration biopsy. There is a substantial percentage of early cancer of the lung in which bronchoscopy will fail as a diagnostic procedure. Aspiration biopsy of selected tumors is a valuable and relatively safe diagnostic procedure. Pathologists familiar with the appearance of the stained smears can make a diagnosis if tumor tissue is obtained.—*Aspiration Biopsy of Tumors of the Lung, L. F. Craver & J. S. Binkley, J. Thoracic Surg., April, 1939, viii, 436.*—(*L. F. B.*)

**Bronchogenic Carcinoma.**—The incidence of bronchogenic carcinoma is high and ought to demand our attention and concern. The aetiology is unknown, but if heredity plays a part it probably requires an irritant to stimulate the growth of an embryonal rest. The symptoms and physical signs of this condition are indefinite, but with the help of X-ray and bronchoscopy the diagnosis should be possible in a large number of cases. In recent years planigraphy has been of help. An over penetrated film or a Potter-Bucky exposure may reveal the true situation when fluid is present. All cases are entitled to bronchoscopy and a biopsy should be made even though these may not exclude carcinoma. The instillation of iodized oil will often demonstrate an occluded bronchus, especially in the upper lobes which are difficult to reach with a bronchoscope. Occasionally thoracotomy and exploration are indicated. Bronchogenic carcinoma must sometimes be differentiated from chronic pulmonary disease, especially pulmonary tuberculosis and lung abscess, and occasionally from atypical pulmonary infarction, pleuritis, lymphoblastoma and fluid cyst. A metastatic process may give rise to the first symptoms, the most common location for these being the skeletal system, the regional lymph nodes and the pleura. Skeletal lesions may be osteoplastic, osteolytic or a combination of the two. The prognosis is bad, most cases succumbing in one or two years. Radiation and surgery are the recognized forms of treatment, but radiation with the present technique is merely palliative while surgery offers hope only in selected cases. Eight cases are reviewed.—*Bronchogenic Carcinoma, W. E. Hawes & S. G. Schneck, Radiology, January, 1939, xxxii, 8.*—(*G. F. M.*)

**Bronchogenic Carcinoma.**—In an attempt to obtain more information about the early manifestations of primary carcinoma of the lung, in the stage when the disease is still amenable to surgery, the case records of 60 patients admitted to the Jewish Hospital of Brooklyn were studied. Forty-seven of these were free from thoracic symptoms until one month before admission and the other 13 gave no history referable to the thorax. A final diagnosis of carcinoma of the lung was made in all these cases. In 13 the diagnosis was confirmed by necropsy; in the remainder the diagnosis rested upon clinical course and X-ray findings alone or combined with biopsy. In 81.7 per cent, the patients were between forty-five and sixty-five years of age. There were 47 males and 13 females. There are no symptoms pathognomonic of carcinoma of the lung. The character of the onset is extremely variable. The disease may start insidiously, simulating a prolonged bronchitis or it may be acute, simulating an acute pneumonic process. Epigastric distress is an important complaint; it was elicited in 10 of the 13 patients who had no thoracic symptoms. This symptom was more commonly present when the left side was involved and the process was basal. Frequently there is associated anorexia, nausea, vomiting, constipation and various vague abdominal complaints. Some of these patients had been treated for months for a supposedly obscure intraäbdominal condition. Of the 47 patients in whom thoracic symptoms were present on admission, cough was present in 91.5 per cent; other frequent symptoms were chest pain, expectoration, dyspnoea, weight loss, haemoptysis and asthmatic wheezing. The general well being of the majority of these patients was noteworthy. Physical signs are extremely variable and at times none can be elicited. Since, in the very early stages, the neoplasm is localized to the bronchus, particular attention should be directed to bronchial signs such as wheeze and prolonged expiratory sound. The persistence of an area of impaired percussion with depressed breath sounds, in the absence of recent infection of the respiratory tract, should be considered as due to carcinoma until proved otherwise. Where the growth is still early and local, the roentgen findings vary with the location of the tumor depending upon whether the latter is hilar, parenchymal or peripheral. In the very early stages, particularly in the hilar type, there may be no roentgen findings. Oblique and lateral roentgenograms are important supplements to the routine postero-anterior projection. Bronchography is also of definite value, while bronchoscopy is the single greatest

available aid in the early diagnosis of lung cancer. When pleural fluid is present, diagnosis is sometimes established by the finding of tumor cells in the aspirated fluid. If the clinical course suggests malignancy, a single negative bronchoscopy or futile search for tumor cells in pleural fluid do not rule out the diagnosis. In some patients the diagnosis may be established by repeating these examinations after some time. In the later stages, biopsy of lesions suspected of being metastases, such as skin nodules or enlarged lymph nodes, will occasionally establish the diagnosis. Exploratory thoracotomy must also be considered as an accessory means of diagnosis; it is particularly valuable and justified as a method of early diagnosis when other methods have failed. When biopsy of representative tissue is not possible during bronchoscopy, diagnosis can occasionally be made by examining aspirated bronchial secretions for tumor cells. When a patient of "cancer age," without apparent cause, develops spontaneous pneumothorax or empyema and fails to rally after the proper therapy is instituted, the diagnosis of carcinoma of the lung must be considered until ruled out. Illustrative case records are cited.—*Early Manifestations of Carcinoma of the Lung, L. A. Hochberg & M. Lederer, Arch. Int. Med., January, 1939, lxiii, 80.*—(*H. R. N.*)

**Pneumonectomy for Bronchogenic Carcinoma.**—Because of the lack of record in the literature of the condition of patients five years after pneumonectomy for primary lung cancer, the author presents the record of such a case, in which after five years, following right pneumonectomy for primary bronchogenic carcinoma, the patient remains entirely free from metastatic disease. In the author's series another patient has lived nearly five years and 2 others nearly three years, such results justifying the statement that the benefit of surgical aid be considered for all such patients if metastatic disease is not demonstrable at the time of clinical discovery of the primary lesion.—*Apparent Cure of Primary Carcinoma of the Lung by Pneumonectomy: Case Report of a Five Year Follow-up Study, R. Overholt, J. A. M. A., August 19, 1939, cxiii, 673.*—(*G. L. L.*)

**Bronchogenic Carcinoma.**—The authors present case summaries of 7 patients to illustrate the difficulties of diagnosis that may frequently confound both the clinician and the radiologist. Carcinoma of the lung not infrequently presents a clinical picture resembling pulmonary suppuration so closely that an exact diagnosis is possible only by bronchoscopy, exploration or autopsy. Even at autopsy a carcinoma of the lung may be overlooked entirely because of the overshadowing features of pulmonary suppuration. Any long unresolved pneumonia or persistent suppuration that does not yield to the usual therapeutic measures should be considered a case of probable neoplasm until proved otherwise. The diagnosis of carcinoma of the lung has increased in twenty-five years from an incidence of 1 per cent to 10 per cent of all carcinomata encountered. When situated in the periphery of the pulmonary field, the first symptoms are referable to the thoracic wall. Effusions should be aspirated, the fluid centrifuged and prepared for microscopical sectioning and study. Pain referred to the shoulder, due to diaphragmatic involvement, may occur, as may also paralysis of the diaphragm due to infiltration of the phrenic nerve. At certain stages in the development of pulmonary carcinoma it may present, owing to inflammation, all the characteristics of a pulmonary abscess, of purulent bronchitis, of bronchiectasis or of tuberculosis. Atypical intrapulmonary lesions resembling infection but not entirely explained on the basis of inflammation are most frequently due to neoplasm.—*Carcinoma of the Lung Simulating Inflammatory Disease, E. Holman & P. Pierson, J. A. M. A., July 8, 1939, cxiii, 108.*—(*G. L. L.*)

**Bronchogenic Carcinoma.**—There is an apparently wide variance in the conceptions of authors as to the histiogenesis and structure of carcinoma of the lung. The available data have contributed materially to our knowledge, but no uniformity has as yet been attained in classifying these growths. The difficulty may be superficial, however, rather than essential. In the authors' opinion a classification on a histogenic basis, combined with a nomenclature derived from the cell making up the growth, rather than from the structure which the cell formed, will go far toward simplification of the problem. All carcinomata primary in the lung, it now seems clear, can be classified into one of three groups—squamous cell, reserve cell and columnar cell. The occasional overlapping of groups is not unexpected, since all carcinomata of the lung, the authors believe, are derived from a common ancestor cell, the reserve cell. Seventy-four cases of primary carcinoma of the lung were encountered in 6,623 autopsies on individuals over one year of age. Males and females were represented in the proportion of 11:1. The age range was from twenty-one to seventy-five years. The average duration of illness was five months. Thirteen patients died in the fifth, 33 in the sixth, and 19 in the seventh decade of life. In almost half of the cases the primary

growth was located in one bronchus or the other. Thirty-seven of the 74 cases were squamous cell, 21 were reserve cell and 16 were columnar cell carcinoma.—*Carcinoma of the Lung, an Analysis of Seventy-Four Autopsies, R. D'Aunoy, B. Pearson & B. Halpert, Am. J. Path., September, 1939, xv, 567.*—(*J. S. W.*)

**Bronchogenic Carcinoma.**—The author presents a study of carcinoma among patients of the Veterans Administration. The total number of primary bronchogenic carcinomata from 1927 to the first six months of 1937 inclusive was 160. All carcinomata found during this period numbered 1,167 and the autopsies performed, 7,398. In studying the figures it was found that there had been a steady rise in the number of all carcinomata, as well as in the bronchogenic type, from 1931 to 1937. The percentage relation of all carcinomata to all autopsies increased by 28 per cent from 1932 to 1937; for the same period the percentage relation of primary bronchogenic carcinomata to all autopsies increased by 120.8 per cent and the relation to all carcinomata by 69.6 per cent. The absolute increase of this type of tumor is unrelated to the increase of carcinoma in general. The factors which may be responsible for this absolute increase are the large percentage of preceding diseases of the respiratory tract and the fact that a large number of the patients were in occupations accompanied by exposure to irritations of the respiratory tract and traumatizations of various kinds. Another factor is the added interest in cancer on the part of the physicians of the Veterans Administration, resulting in an increase of the number of pulmonary cancers diagnosed both antemortem and postmortem. Another factor responsible for this relative increase in incidence is the entrance of the veterans' group into the cancer age period.—*The Incidence of Primary Bronchiogenic Carcinoma, P. B. Matz, J. A. M. A., December 3, 1938, cxi, 2086.*—(*G. L. L.*)

**Dermoid Cyst of Mediastinum.**—A large dermoid cyst was removed surgically from the anterior mediastinum of a fifty-four year old female who gave a history of gastric distress and postprandial vomiting of twenty years' duration, and more recently, sudden severe epigastric pain and profuse haematemesis. X-ray studies of the oesophagus, stomach and duodenum were negative. An X-ray film of the chest, however, revealed a uniformly opaque mass adherent to the left cardiac border. Aspiration biopsy was unsuccessful. The postoperative course was uneventful and the gastric symptoms diminished markedly. In the absence of roentgen evidence of oesophageal compression, the gastric symptoms are attributed to reflex vagal irritation and the haematemesis to rupture of an oesophageal varix.—*Hématèmèse et kyste dermoide du médiastin, R. Lemieux, J.-E. Perron, H. Nadeau & G. Drouin, Laval méd., January, 1939, iv, 37.*—(*A. B. C.*)

**Mesothelioma of the Pleura.**—Primary malignant neoplasms of the pleura are very rare. From a study of the literature only 14 cases in 20,829 necropsies were found. The symptoms are those of massive pleural effusion. Metastases may occur and the patient will usually die within a year or so of the onset. The diagnosis may be made certain only by microscopical examination of the pleura, although it may be suspected from the insidious course, the finding of thickened pleura and fluid in the chest. The only treatment available is the palliative course of repeated aspiration of pleural fluid in order to relieve dyspnoea. The case of a thirteen-year old school boy is reported. This patient died ten weeks after onset of the disease. At autopsy the left pleural cavity was found to be very large and filled with 2 litres of serosanguineous fluid. The pleura had been entirely replaced by oedematous grayish-white neoplastic tissue. The left lung was atelectatic. The mediastinal lymph nodes on the left side were enlarged. The neoplastic tissue appeared to have infiltrated the pericardium and perforated the diaphragm. Microscopically the tumor was composed of cells varying in shape from round to a thick spindle, with large hyperchromatic nuclei and a relatively small amount of cytoplasm. A diagnosis of primary mesothelioma of the left pleura with metastases to the pericardium, diaphragm and lung was made.—*Primary Mesothelioma of the Pleura, G. L. Birnbaum, Quart. Bull. Sea View Hosp., July, 1939, iv, 462.*—(*A. A. E.*)

**Adenoma of Bronchus.**—Benign tumors of the bronchus constitute about 6 per cent of all endobronchial neoplasms according to Morlock and Pinchin; of the benign tumors adenoma is the most common. The patient whose history is reported was a forty-eight year old housewife whose symptoms began ten years before admission; at that time she had had pneumonia in the right lung and empyema. Five years before admission she had a sudden massive haemoptysis and continued to have large haemoptyses at intervals of three to five months. She had no cough, expectoration, fever or night sweats. Roentgenograms demonstrated a

paramediastinal triangular shadow in the right lower lobe with thickening of the right pleura and retraction of the heart and mediastinum to the right. This was interpreted as atelectasis of the right lower lobe. Bronchoscopy disclosed a purplish red mass about 1 cm. below the carina arising from the mesial and anterior wall of the right main bronchus and almost completely occluding its lumen. Bioptic examination showed a connective tissue stroma with a matrix of cuboidal epithelial cells of a glandular type. The epithelial cells had a definite regular alveolar arrangement and there was no evidence of malignancy. The mass has been repeatedly treated by electrocoagulation through a bronchoscope. Subsequent bronchograms showed bronchiectasis in the right lower lobe. Benign adenomata generally cause symptoms before the age of forty and occur more frequently in women than in men. The onset is insidious and earliest symptoms may be nothing more than an occasional cough. As the tumor enlarges the patient develops an irritant cough, dyspnoea when in certain positions, and haemoptyses of sudden and alarming character. Roentgenograms show no abnormalities until the tumor is large enough to occlude a bronchus and produce atelectasis in the corresponding portion of the lung. Grossly, bronchial adenomata are sessile or pedunculated tumors which bleed easily on manipulation. Microscopically they are covered by a smooth layer of epithelium beneath which is a dense layer of fibrous tissue and beneath this layer are nests of epithelial cells with small round or oval nuclei which may have an alveolar arrangement. Bronchial adenomata do not metastasize. Treatment is best carried out by electrocoagulation through the bronchoscope and the prognosis is good if a diagnosis has been made and treatment instituted before suppurative pulmonary complications have occurred.—*Adenoma of the Bronchus: Report of a Case with Clinical Review, I. G. Tchertkoff & E. Klosk, Quart. Bull. Sea View Hosp., January, 1939, iv, 202.*—(*A. A. E.*)

**Pneumonectomy for Tumors of Lung.**—In the treatment of malignant tumors of the lung lobectomy has been uniformly unsatisfactory owing to local recurrences. In the majority of cases it is necessary to amputate the primary bronchus close to the trachea in order to circumvent the tumor. This can be accomplished only by total pneumonectomy. The operation which the author describes is termed "graded pneumonectomy" because it permits the interruption of the operation at one or several points when the condition of the patient does not warrant a complete one-stage procedure. Prior to the operation, pneumothorax of the affected lung is attempted in all cases and was accomplished in the vast majority of the cases which form the basis for this report. It is much easier to remove a collapsed lung than an inflated one and at the same time the pneumothorax helps to prepare the patient for the physiological effects of the loss of one lung. Following the successful collapse of the affected lung, 50 cc. of beef broth was routinely injected in the pleural cavity on the pneumothorax side. This injection, given forty-eight to seventy-two hours before the operation, serves to protect the patient from later infection and also acts as a stimulus to the formation of granulation tissue, the exudation of plasma and the obliteration of the dead space which will result from the removal of the lung. This injection of broth causes a brief constitutional reaction with a mild fever as the effusion develops. The thoracic cavity is shortly lined with fibrin which later is largely replaced by granulation tissue. Following the operation there is an accumulation of serosanguineous fluid which seems to have an inhibitory effect on bacterial growth, for empyema did not develop in any of the cases. The fibrin present forms a scaffold for the ingrowth of fibroblasts and endothelium. This finally results in the formation of a multilobular cystic fibrous body which occupies the previously empty thoracic space. The most satisfactory anaesthesia was found to be a combination of nembutal and cyclopropane, the latter delivered from a closed gas circuit through a tight-fitting mask. As the method of closure of the bronchial stump determines the success or failure of a pneumonectomy, the procedure for ligating the primary bronchus is given in detail. Access to the hilum of the lung is obtained by the anterior approach, the incision being made over the third intercostal space anteriorly. The ribs are not resected but are forced apart by a "rib-spreader" with resulting disarticulation of the third and fourth costochondral junctions. With the exposure of the hilum the pulmonary artery is first ligated. The primary bronchus is then tied near the bifurcation of the trachea above the point at which the bronchial artery enters the posterior bronchial wall. The pulmonary veins are next ligated, followed by amputation of the main bronchus. If the operation is to be in two or more stages the ligatures around the pulmonary veins are left untied for later approach. An important feature of the operation is the covering of the bronchial stump with a pedicle flap. Early in the

operation the phrenic nerve is crushed with a resultant temporary elevation of the diaphragm and partial obliteration of the thoracic cavity. In 20 patients total pneumonectomy was successfully performed by the technique described. There were 2 deaths in the series, one in fourteen days, from a failure of the peripheral circulatory system (delayed shock?) and the other, one week after the operation, from coronary disease not previously recognized. In both instances the bronchus had healed completely and there was no infection of the pleural cavity. These 20 cases represented a favorable group in which the hilar structures were free from disease and the lung was in the main free from the chest wall. Great care should be exercised in the closure of the bronchial stump and the placing of the sutures therein, but the keystone to secure occlusion of the bronchus and its subsequent healing, and likewise to the success of total pneumonectomy, would seem to be the primary encircling ligature placed around the primary bronchus just distal to the bifurcation of the trachea.—*Graded Pneumonectomy in the Treatment of Tumors of the Lung, W. F. Rienhoff, Jr., Bull. Johns Hopkins Hosp., March, 1939, lxiv, 167.*—(*J. S. W.*)

**Closure of Bronchus.**—The method of a two-stage total pneumonectomy is outlined and may be used when the necessity for such a procedure arises. The bronchus, whether right or left, is cut across in the mediastinum not far from the bifurcation of the trachea. The incision is made slightly oblique to the long axis of the bronchus, on a line running from above and lateral to below and medial. In addition, the cut is made on the bias so that the posterior membranous portion is a trifle longer than the more anterior cartilaginous wall. Interrupted fine "a" silk sutures are placed in the membranous portion in such a way that a portion of the posterior membranous tissue is picked up a few millimetres proximal to the cut edge. The needle is then inserted into the cut edge of the mucous membrane and the cartilaginous ring of the bronchus directly opposite. From ten to twelve such sutures are placed in a fan-shaped pattern. When the knots are tied, the relaxed and relatively tough posterior membranous wall is snugly fitted to the inner surface of the semicircular cartilaginous ring and, as a result of the way the sutures have been placed, the membranous portion is rolled over the cartilaginous cut edge in a way corresponding to the inversion of the intestinal wall. There is no tension on the suture line. Two parallel rows of through-and-through mattress sutures are placed proximal to this interrupted row. They are separated from the end and from each other by the width of one cartilaginous ring and the sutures pass completely through the bronchial walls. For these sutures no. 1 chromic catgut is used. The membranous end of the bronchus is abundantly supplied with blood by the bronchial artery. By using an oblique incision and a similar technique for closure, an adjacent portion of the trachea may be removed with the involved bronchus.—*A Two-Stage Operation for Total Pneumonectomy in the Treatment of Carcinoma of the Lung, Demonstrating a New Technique for Closure of the Bronchus, W. F. Rienhoff, Jr., J. Thoracic Surg., February, 1939, viii, 254.*—(*L. F. B.*)

**Pneumonoconiosis.**—Recent clearly defined concepts of pneumonoconiosis are to-day becoming again confused because enthusiastic observers in many fields are drawing deductions without sufficient knowledge of the problem as a whole. Animal experimental work is offering the best solution to the crystallizing of correct pathological concepts of the reaction of various dusts upon body tissue, and injection methods are becoming more valuable and time saving than inhalation experiments. The author discusses the aetiology of the condition from three viewpoints; first, the irritant capacity of different minerals; second, factors governing the behavior of air-borne dust; finally, the host factors that modify the cellular reactions. The minerals are classified by groups into free silica, combined silica, nonsiliceous particles and mixtures of free silica and other substances. His observations are largely the results of his animal work with intravenous injection of minerals. All the crystalline and cryptocrystalline forms of free silica are found to be as active as or more active than the standard normal quartz. All of them have also produced progressive fibrosis of a silicotic type in guinea pigs, rabbits and white rats. The observations on the amorphous varieties of free silica are more variable. Colloidal silica is exceedingly irritating and in too large an intravenous dose it causes almost immediate death. If the same suspension is allowed to gel, the silica promptly loses its irritating properties. The natural minerals are much more active. Pure diatomite produced a reaction which was indistinguishable from that to quartz, while opal caused a nonhyaline fibrosis which was atypical and which had begun to retrogress after a period of one year. So far, none of the silicates have proved capable of producing a fibrous type of reaction. Beryl is apparently

toxic and produces acute local inflammation that invariably leads to death. A few of the silicates, notably biotite mica and some of the clays, have provoked local proliferation of mononuclear phagocytes. Whether this early reaction will ultimately end in fibrosis awaits demonstration. If fibrosis should develop it will do so only after much longer contact than is required in the case of free silica. Synthetic sodium silicate is exceedingly toxic when injected, but probably any quantity which could be inhaled would be harmless. The asbestos group of silicates, including anthophyllite, amosite, amphibole, crocidolite and chrysotile, have invariably failed to cause fibrosis or even early proliferation either intravenously in rabbits or in the peritoneal cavity of guinea pigs. These observations are at variance with the well recognized capacity of asbestos to produce fibrosis of the lungs. A considerable number of observations are pointing to the hypothesis that the fibrous silicates behave in a different manner from the particulate minerals and that they irritate the lungs mechanically. Unlike quartz and the other active free silicas, not only have they failed to produce fibrosis in organs other than the lungs but they have not become more irritating as particle size decreases. In experimental animals the fibrosis of pulmonary asbestosis does not progress after exposure to the dust is discontinued, as invariably happens with free silica. The absence of asbestosis bodies in tissues other than the lungs suggests that chemical changes peculiar to that organ are responsible for their development. It has been suggested that the irritated tissues attempt to protect themselves by laying down the coating on the fibres. Subcutaneous injection of asbestosis bodies recovered by digesting human lung tissue with antiformin has caused no fibrosis in guinea pigs. By any of the injection methods carborundum, or silicon carbide, has failed to cause even a suggestion of fibrosis. This constitutes very good evidence for rejecting the properties of hardness and sharpness as a cause of such reaction. None of the nonsiliceous minerals experimented with have caused primary fibrosis. A few have caused slight chronic inflammatory reactions. Some have been toxic. Diamond, the hardest known mineral, and aluminum oxide, a common abrasive, are inert. Soft coal is more irritating than anthracite. Injections indicate that the presence of various other minerals in a mixture may retard the usual reaction to silica, restricting the extent of tissue involvement and modifying the form of the cellular response. The experiments suggest that inhibition is most effective in the presence of small amounts of silica. The mechanism by which inhibitors exert their effects has yet to be demonstrated. Many attempts have been made to evaluate the solubility of silica. Actual demonstration of solution of particulate silica in the living body is difficult. At the present time it is not generally conceded that the excretion of silica in the urine is of particular aetiological or diagnostic significance. It is probable that if the silica is dissolved most of it combines with the tissues in the immediate vicinity of the particles and that the soluble silica demonstrated in the urine is very largely derived from different foods. In the Saranac Laboratory the amount of soluble silica has been determined colorimetrically on all the physiological salt solution suspensions used for testing animals. There is apparently no parallel between the capacity of a siliceous mineral to produce reaction and its degree of solubility. If test tube experiments parallel conditions in the body, it would seem that some other factor besides solution in water is essential to the production of silicosis. Such factors as the size, shape and specific gravity of particles and the factor of agglutination affect the proportions of silica atmospheric suspensions. The protective action of the upper respiratory tract is also of importance, although fibrous material is not so easily handled as large particulate material. Variations in the pathological response to inhalation of mixed dusts in different industries are no doubt due to two sets of factors, the one acting outside the body to reduce the amount of silica inhaled, the other inside the lungs to delay and modify the irritating effects of silica. Evidences of inhibitory action are not confined to experiments on animals; in industrial plants, reductions in the proportion of inhalable atmospheric silica have been repeatedly demonstrated and delayed responses to silica in mixed dusts are not uncommon. Under former conditions of employment only about one-fourth of any group of miners of hard rock would develop silicosis while the other 75 per cent seemed to be immune. There is as yet no proof that the tissues of different normal persons vary in their capacity to react to irritating minerals. With the large doses of free silica employed for the injection tests every animal in any series has exhibited a similar response. The factor of infection is the cause for variation in susceptibility which has been studied most intensively. In the case of the lungs this complication may produce anatomical alterations which mechanically influence the deposition of inhaled dust, and it may possibly act in more subtle ways to alter the tissue responses to

certain kinds of dust. The more subtle influences of infection are not well understood. The presence of chronic tuberculous infection complicated by long continued inhalation of silica dust seems to result in more accentuated fibrosis than develops with either infection or dust inhalation alone. Experimentally, the only silicate dust which has produced this exaggerated fibrosis in the presence of infection is chrysotile asbestos.—*Etiology of Pneumoconiosis, L. U. Gardner, J. A. M. A., November 19, 1938, cxi, 1925.*—(*G. L. L.*)

**Silicosis.**—The case report is deemed noteworthy because of the absence of any tuberculous infection and because of exposure to very pure silica dust. The patient, a forty-two year old Scotchman, operated a sandblast in a foundry from 1924 until 1929. The work was done without a helmet and there was no adequate means of dust removal. In 1929 he developed cough and sputum, accompanied by increasing dyspnoea. The symptoms increased and he died two years after onset of symptoms. Sputa were persistently negative for tubercle bacilli on direct smear and concentration. Roentgen films of the chest showed dense hilar shadows with nodular shadows throughout both lung fields. At autopsy there was no evidence of tuberculous involvement. Microscopically the outstanding features were the relatively small number of air-containing alveoli due to the presence of an inspissated exudate, the lining of the alveolar walls with a continuous layer of flattened cells, and the virtual restriction of the fibrosis to the interlobular septa and the visceral pleura. In some areas the exudate was finely granular, eosinophilic and acellular; these areas showed doubly refractive clear crystals thought to be cholesterol. In other areas this exudate was composed of degenerating spherical cells, in the cytoplasm of which fat and dust particles could be seen. Throughout this exudate the acid treated incinerated sections revealed the presence of scattered anisotropic mineral particles distributed in a fairly uniform manner. The incinerated sections of fibrous tissue showed the presence of large amounts of finely particulate siliceous material, varying from 3 $\mu$ to 7 $\mu$ in their greatest diameter. Chemical analysis revealed the total silica content to be 1.31 per cent of the dry lung tissue and nearly 35 per cent of the complete lung ash. The material used in the sandblasting was obtained from the St. Peter sandstone formation in the upper Illinois valley. This sandstone is characterized by a striking uniformity of quality and is of unusual purity. The lung ash contained 95.16 per cent silicon dioxide while the sand itself contained 99.01 per cent. This slight difference was to be expected as small amounts of various silicates are found in the lungs of all adults. The percentage of quartz found in the lung ash in this case is unusually high. It is interesting that the heart was not enlarged nor did it present any evidence of damage. The finger nails were curved but there was no clubbing. This can probably be accounted for by the absence of infection.—*A Study of a Fatal Case of Uncomplicated Silicosis, A. R. Riddell, C. M. Jephcott & D. A. Irwin, J. Indust. Hyg. & Toxicol., November, 1938, xx, 566.*—(*H. R. N.*)

**Anthracosilicosis and Heart.**—Congestive heart failure is a common occurrence in cases of far advanced anthracosilicosis. Because of the difficulty in determining at the time of admission whether the common symptom of dyspnoea is due to cardiac insufficiency or to the anthracosilicosis, the authors have used the electrocardiograph as well as clinical and laboratory studies to detect early pathological changes in the heart. Of 25 miners studied, 16 had anthracosilicosis without pulmonary tuberculosis. Nine had either moderately advanced or far advanced pulmonary tuberculosis in addition to anthracosilicosis. The duration of occupation as miners ranged from three to forty years. Roentgenograms of the heart showed that 2 had cardiac enlargement. Secondary polycythaemia and reduction of vital capacity were frequent occurrences. No appreciable variation from the normal venous pressure and pulmonary and complete circulation time was noted. The electrocardiograms in the majority of instances showed myocardial damage. Right axis deviation occurred in only 3 cases, left axis deviation in 10, and no deviation in 12 cases. Coronary occlusion was present in one case. Functional tests showed that practically all the miners studied had abnormal acceleration of the pulse rate after slight exertion.—*The Heart in Anthracosilicosis, J. F. Giering & R. Charr, J. A. M. A., August 12, 1939, cxiii, 574.*—(*G. L. L.*)

**Pneumothorax in Anthracosilicosis.**—To determine the frequency of spontaneous pneumothorax as a complication of pneumonoconiosis, the authors reviewed the records of 507 anthracite coal miners incapacitated by chronic pulmonary disease. All these men had anthracosilicosis; 308 had coexisting pulmonary tuberculosis and one had metastatic pulmonary carcinomatosis. Pneumothorax occurred spontaneously in 22 (4.3

per cent) patients. Among the 199 patients with simple anthracosilicosis it was found in 7 (3.5 per cent). Among the 307 patients with coexisting tuberculosis it was seen in 14 (4.5 per cent); it also occurred in one instance in association with pulmonary metastasis (0.3 per cent). The ages of the patients ranged from twenty-five to fifty-two years and they had been exposed to mine dust for periods ranging from three to thirty-six years. Eight patients had second stage and 14, third stage anthracosilicosis. In 3 instances the pneumothorax was bilateral, in 19, unilateral. Of the 7 patients with uncomplicated anthracosilicosis, one died. Of the 15 with a complication, the patient with carcinomatosis and 10 of the 14 with tuberculosis died. Emphysema, pleural adhesions, tuberculosis and the accompanying severe cough favor the development of pneumothorax in anthracosilicosis. The effects of these forces are largely overcome by thickening of the visceral pleura or obliteration of the pleural cavity, practically constant features of anthracosilicosis.—*Spontaneous Pneumothorax in Anthracosilicosis, M. J. Sokoloff & J. T. Farrell, J. A. M. A., April 22, 1939, cxii, 1564.*—(*G. L. L.*)

**Silicosis in Dentistry.**—The author reports a dental technician who worked for nineteen years at the same operation, the polishing of dentures. After the development of cough and progressive dyspnoea a diagnosis of pulmonary tuberculosis was made, and later because of the typical X-ray appearance of nodular densities from apex to base, further investigation substantiated a diagnosis of silico-tuberculosis. The patient died five months after admission to the hospital, and about two years after the beginning of symptoms. Postmortem examination confirmed the diagnosis. The total silica of ash of the dried lungs was 10.1 per cent. The patient allegedly was using pumice in his work, but since there is considerable doubt that pumice contains free silica in its composition, a sample of the material used by the patient was analyzed, and it was discovered that the abrasive was a less expensive substitute called "pummy," and contained 48 per cent free silica. This substitute "pummy" is apparently sold to New York dental laboratories in the same amount as pumice, and obviously constitutes an occupational hazard to workers in mechanical dentistry. The question of the possible harmfulness of pumice must await further investigation. In the meantime, "pummy" should be discarded as dangerous abrasive material, and polishing apparatus should be equipped with exhaust hoods.—*The Silicosis Hazard in Mechanical Dentistry, L. E. Siltzbach, J. A. M. A., September 16, 1939, cxiii, 1116.*—(*G. L. L.*)

**Dyspnoea in Silicosis.**—Hitherto the dyspnoea of silicosis has been theoretically explained by the two factors of nodulation and alveolar consolidation. Dyspnoea is frequently present, however, in the absence of nodulation. The authors have made extensive microscopical studies of the pathological changes of acute silicosis, and discuss the three recognized and described types of silicosis, namely peribronchial silicosis, nodular silicosis and acute silicosis, the latter type providing the key to the authors' explanation. Sections of the lung all the way from hilum to pleura, stained with Masson's trichrome stain, were most valuable in this study. Black and white photomicrographs were useless, and haematoxylin and eosin stain was also unsatisfactory. Such sections show three general manifestations of morbid change: avascular areas, overvascular areas and a region of invasion. The region of invasion, where collagen constricts the capillaries, is the most important region for intensive study. In the avascular portion, capillary occlusion by external pressure causes a traffic jam of red blood cells, with a damming back of the blood into the arterioles and a lack of drainage of the veins. The blood in the larger vessels in the avascular areas disintegrates. Overvascular areas, where there is immense dilatation of the capillaries and engorgement of the veins and arteries with viable red blood cells, seem to compensate for avascular regions in different portions of the section. In view of these observations the authors are convinced, first, that nodulation is not the prime factor in silicotic dyspnoea, and, second, that capillary obstruction, either as an entity or combined with alveolar consolidation, as observed in acute silicosis or as an acute manifestation of the late stages of chronic silicosis, is the chief pathological factor which causes dyspnoea. The dyspnoea of silicosis is principally due to occlusion of pulmonary capillaries in large, localized regions of the lung, with compensating capillary dilatation in other regions. In cases of acute silicosis capillary occlusion is spectacularly present. Circulatory changes seem to have played an important part in one way or another in practically all the cases observed in these studies.—*Dyspnea of Silicosis: What Causes It?, L. G. Cole & W. G. Cole, J. A. M. A., September 13, 1939, cxiii, 1216.*—(*G. L. L.*)

**Tuberculosis and Pneumonoconiosis.**—Silicosis, in the absence of an infective

process, is a benign condition, and frequently allows its victims to reach old age. Asbestosis makes more serious and rapid inroads into the functional efficiency of the respiratory system, but death is frequently caused by infectious processes either tuberculous or nontuberculous. The problem can still be resolved into "the seed and the soil," silica or silcates in the lungs acting as a fertilizer for the propagation of tubercle bacilli. The incidence of tuberculosis is likely to be greater among workers in fixed or confined work places than among those in the open. The incidence of tuberculosis among grinders shows two peaks, one in the early adult period and a sustained peak between the ages of thirty-five and fifty-five. Among coal miners there is a peak at the early adult period while among stone masons the peak is at middle or later life. The absence of a middle age peak among coal miners shows that their occupation does not adversely affect them with regard to tuberculosis in spite of the pneumonoconiotic risk. The young adult peak among grinders is high and is due to repeated exposure to infection accompanied by inhalation of silica dust. Studies show that more tuberculosis is found in women than men in the pottery and asbestos industries. The leading symptom of uncomplicated silicosis is dyspnoea on exertion. Cyanosis is not common except in advanced cases. In asbestosis dyspnoea on exertion is frequently accompanied by cyanosis. Rapid loss of weight suggests the appearance of an infective process, usually tuberculosis. In the young adult group, acute or subacute cases of silico-tuberculosis do badly unless the exudative phase can be overcome before anatomical damage is excessive. Early or more chronic cases may do well. The general characteristic of the disease in the middle-aged group is chronicity although recovery or permanent arrest is not common. In later life may be found the terminal stages of disease acquired in middle life, or acute tuberculosis on a background of tuberculosis. Collapse therapy is contraindicated unless the lesion is definitely unilateral.—*Tuberculosis in Relation to the Pneumonokonioses, H. M. Turner, J. Roy. Inst. Pub. Health & Hyg., June, 1939, ii, 381.*—(*L. F. B.*)

**Silicosis in British Coal Fields.**—Anthracosis, colliers' asthma or colliers' phthisis has been known for a hundred years as a disabling industrial pneumonoconiosis. The term "anthracosis," denoting black lung, is a misnomer when applied to lesions due to other factors, that is, silica dust, and a better term is silico-anthracosis, or anthracosilicosis, as used in the United States. There are striking variations in the number of cases in different fields, measured by the number of certificates issued by the Silicosis Board in respect of death, partial and total disablement. All fields north of South Wales are considered as the Northern Coalfields. Among a total of 488,567 employees for the period 1932–1936, 22 deaths were certified and 74 partial or total disablement certificates issued. The Southern Coalfields include South Wales, Monmouth, Forest of Dean, Somerset and Kent. Here, among 127,273 men employed for the same period there were 210 deaths from silicosis and 997 cases of total or partial disablement. This marked disparity requires explanation. In the Northern fields the coal is bituminous and the incidence of silicosis falls almost entirely on those engaged in pit-sinking and tunnelling, in which sandstone beds are not infrequently met with. In the Southern group both anthracite and bituminous coal are mined, the carbon content is higher, and free silica is constantly present in the air coming from the drilling places whose roof consists of quartzite rock. Figures are shown illustrating typical seams. In South Wales the roof is largely quartzite rock, often resting immediately on the coal seam. Dust from the roof is inevitably carried by drilling or blasting operations into the air of the working place. Shales, mudstones and fireclays are sedimentary rocks of low total silica content (20 to 60 per cent) and the free silica is small and loosely set in the form of quartz grains. Lancashire shale has a total silica content of about 31 per cent, mostly secondary white mica. Southern "hard headings" are mostly quartzite, angular quartz grains welded by siliceous cement and also containing mica, siderite, calcite and rutile. The total silica is 90 per cent or more and free silica 70 to 80 per cent. Ninety per cent of dust found in lungs is less than 5 micra in diameter, most of it between 0.1 and 3 micra. The dust consists of insoluble or nearly insoluble particles of anthracite, sericite, rutile, muscovite, epidote, calcite, siderite, limonite and quartz and in the case of bituminous coal, vitrain, fusain, clarain and durain. A few large particles reach the bronchioles and alveoli. Anthracosis and asbestosis bodies are occasionally found even in normal lungs. Components of mine dust which are of interest are sericite and quartz. The former is a secondary white mica, a hydrated silicate of aluminum and potassium and occurs in abundance in shales, mudstones and fireclays, also in mine dust during drilling and blasting operations. W. R. Jones in 1933 propounded the theory

that it was the cause of silicosis. Coal miner's lung ash was found to contain numbers of sericite needles, especially numerous in silicosis, as shown by polarized light. However, these lungs contained also far more minute quartz particles not thus shown. Experimentally, it has been impossible to produce pulmonary fibrosis by dusting with sericite whereas control animals dusted with quartz developed it readily. Clinically, silicosis occurs in such occupations as flint-grinding where no sericite is found. It is the consensus that silicosis is due to inhalation of free silica. Two distinct types of silicosis are described. (*1*) That occurring in tunnellers who have drilled through beds of sandstone or quartzite rock. This type is like that of stone masons except that the onset may be more acute. The nodules are discrete and the X-ray films show disseminated nodular deposits. There is a very definite liability to tuberculosis. (*2*) Silico-anthracosis, giving the hard black lung of the consistency of hard rubber. This type develops slowly over twenty to thirty years, is due to working in combined coal and silica dust, and tuberculosis is a less common complication. Research is still continuing in the Southern coalfield which seems to be comparable in many respects with those in Pennsylvania in the United States. Dust counts and petrographic analyses have been made by R. R. Sayers of the United States Public Health Service and 2,711 men were examined clinically and roentgenographically. Except in rock workers, less than 2 per cent of miners developed the disease in a period of less than fifteen years, regardless of the quantity of dust. Of those exposed fifteen to twenty-four years, less than 5 per cent developed the disease with dust of less than 5 per cent free silica content, 14 per cent where the count was 100 to 200 particles per cubic foot, 29 per cent where 200 to 300, and 58 per cent where more than 300. The figures increased with longer employment. The incidence of pulmonary tuberculosis among workers below thirty-five was slightly less than that of the adult male population, but from thirty-five to forty-four it doubled and from forty-five to fifty-four reached five times and fifty-five to sixty-four ten times that of the general rate. Thus, to produce silicosis, the dust must be active, sufficiently small in size, it must be inhaled in sufficient quantity over a sufficient period of time. In South Africa the incidence has been greatly lessened by (*1*) adequate ventilation of working places; control at source by exhaust ventilation is best; (*2*) wetting the rock before loading and in any dusty process; and (*3*) application of dust traps to drills. In blasting, mist projectors should be used. Where the mine roof is chiefly quartzite, blasting should be done only at the end of working shifts. (*4*) The use of efficient masks by workers. The best now have a small tube connected with compressed air, thus providing positive pressure about the nose and mouth. (*5*) Careful examination prior to employment, with elimination of those with either healed or active tuberculosis or subject to attacks of pneumonia; (*6*) frequent dust counts to check effectiveness of prophylaxis.—*Silicosis in the British Coalfield, W. E. Cooke, Tubercle, April, 1938, xix, 289.*—(*A. P.*)

**Cavities in Silicosis.**—*Case Report:* The case here reported was observed at the National Social Insurance Institute, near Budapest, Hungary and was complicated by cavitation. The patient was forty-six years old, a sand-moulder for thirty-four years, and his clinical symptoms noted were dyspnoea, bronchitis and emphysema. The X-ray picture showed a combination of pathomorphological elements characteristic of silicosis. There was a network of cavitations in the left upper lobe. Sputum was consistently negative for tubercle bacilli, even by repeated animal inoculation. Noteworthy is the presence of extensive cavitation without any demonstrable tubercle bacilli. This finding is fairly common. However, the coexistence of tuberculosis is assumed. Cavities develop by the action of silica on the pulmonary tissues and the densities are due to intense inflammatory reaction. Classical cases have been reported from the South African gold mines. —*Cavernous Silicosis, S. Puder, Tubercle, August, 1938, xix, 511.*—(*A. P.*)

**Acid-Insoluble Matter in Sputum.**—Six hundred cc. of sputum were divided into aliquots of 75 cc. and to each varying amounts of finely divided quartz were added. There was approximately 99 per cent recovery using the following method: 10 cc. of concentrated sulphuric acid, 15 cc. of concentrated nitric acid, and 1 cc. of saturated boric acid solution are added to a measured volume of sputum (100 cc.) in an Erlenmeyer flask. The sputum is evaporated by gentle heating until it blackens, and digestion carried to an end by the addition of more nitric acid, drop-wise, as necessary. The clear liquid is transferred to a 15 cc. tube and centrifuged for one hour at 2,000 r.p.m. and carefully decanted. The residue in the flask is quantitatively transferred to the centrifuge tube with water washings, using a rubber policeman. The tube is again centrifuged and the super-

natant fluid decanted. Ten cc. of fresh aqua regia are added to the residue in the tube and allowed to stand overnight. It is then washed twice with water and once with 95 per cent alcohol by repeated centrifuging and decanting. The alcohol washed residue is suspended in a few cc. of alcohol, transferred to a weighing vial, and dried to constant weight in a vacuum desiccator. The weighed residue may be suspended in a few drops of alcohol and examined microscopically under polarized light. The sputa of 83 nonsilicotics, 32 silicotics, and 9 possible silicotics were analyzed by this method. In the 83 nonsilicotics, 96 per cent gave values less than 2.5 mg. residue per 100 cc. of sputum. In 32 silicotics, 84 per cent gave amounts higher than 2.5 mg. per 100 cc. of sputum. Eight silicotics gave amounts ranging from 11 to 21 mg. Some of these had been away from their occupations as long as twenty years. Analysis of the residues with hydrofluoric acid after ignition showed an average of 71 per cent silica. The principal lines in the spectrograph of some of the residues were due to silicon, magnesium and phosphorus. There were also traces of calcium, iron, aluminum, copper, and titanium. The studies indicate that quantitative determination of insoluble matter in sputa of suspected silicotics is valuable diagnostically. Tentatively, 2.5 mg. per 100 cc. of sputum has been chosen as the best dividing line.—*Determination of Acid Insoluble Matter in the Sputa of Silicotics, H. Civin, I. A. Schreier, G. Martello & J. D. Hardy, J. Indust. Hyg. & Toxicol., January, 1939, xxi, 1.*—(*L. F. B.*)

**Ventilation in Granite Industry.**—Studies have shown that the men engaged in drilling operations are subjected to a definite dust hazard. Of the three general types of drilling, men operating the leyner drills are subjected to the greatest hazard. Several types of exhaust ventilation units have been developed that have proved satisfactory in reducing the dust count well below 10 million particles per cubic foot. In the Barre area of Vermont, studies have shown that in the granite cutting and finishing sheds, little, if any, lung changes can be demonstrated roentgenologically in men who have had prolonged exposure to dust concentrations of 10 million particles per cubic foot or less. Ventilation equipment has been extensively installed and dust counts have shown a marked reduction in the number of dust particles per cubic foot. Various types of exhaust hoods are described. Engineering work has shown that the safe upper limit of dustiness, 10 million particles per cubic foot, can be attained.—*Ventilation in the Granite Industry, E. C. J. Urban, J. Indust. Hyg. & Toxicol., March, 1939, xxi, 57.*—(*L. F. B.*)

**X-ray Diffraction in Pneumonoconiosis.**—The failure of chemical analysis methods to establish the presence or absence of pathological levels of silicon dioxide in tissues and fluids in cases of silicosis makes necessary a new approach to the problem in order to differentiate the harmful from the relatively harmless forms of silica. The authors present a study of X-ray diffraction analysis in a group of 5 controls, 14 patients with silicosis and 13 with borderline disease. For the study, thin samples are prepared by packing the powdered tissue into disks from 0.2 to 0.3 mm. in thickness. The X-ray beam, collimated by two 0.01 inch pinholes, is supplied by a Philips metalix tube with a copper target, operating at 20 milliamperes and 27 kilovolt peak. The film is held in a flat holder at a distance of 5.0 cm. from the sample. The exposure time is usually seven hours. Nearly all crystalline compounds give patterns distinctly different from all others. Hence the X-ray method gives definite information as to the identity of the crystalline substances present. Furthermore, a comparison of the patterns with regard to the nature of the lines indicates that there is a variation in size of particles from case to case. Another advantage of the X-ray method is that the tissue can be used for the analysis unmodified by chemical treatment. A further feature is the great sensitivity of the method. With the present technique the authors are able to detect quartz in a concentration as low as 0.2 per cent. This concentration has been found to constitute the threshold of the pathological level. The method should be of value in medico-legal work whenever difficulty is experienced in determining the presence or absence of silicosis.—*X-ray Diffraction Analysis as Applied in Pneumoconiosis, H. C. Sweany & Rosalind Klass, J. A. M. A., February 18, 1939, cxii, 610.*—(*G. L. L.*)

**Tomography in Silico-tuberculosis.**—Tomography can be very useful in distinguishing tuberculous from silicotic pulmonary lesions. By planigraphic analysis of various layers, misleading shadows can more easily be distinguished. This is true especially in those cases where fibrosis and emphysematous blebs resemble tuberculous cavities. Also in cases of silicosis, associated with extrapulmonary tuberculosis, tomography presents important findings by either excluding or confirming pulmonary

tuberculosis. Fibrotic lesions in uncomplicated silicosis yield frequently to tomographic visualization. Late secondary lesions, emphysematous blebs, involvement of the blood vessels, lymphatics, the diaphragm and mediastinum can especially well be demonstrated. Tomographic examinations are of no value in cases of early silicosis. —*Was leistet das Röntgenschichtverfahren in der Begutachtung und Bekämpfung der Silikose und Silikotuberkulose?, W. Silberkuhl & W. Müller, Fortschr. a. d. Geb. d. Röntgenstrahlen, March, 1939, lix, 233.* —(*F. G. K.*)

**Asbestosis.**—Examination of 541 workers in the asbestos textile industry in North Carolina revealed asbestosis in a considerable proportion. The incidence rose with increasing length of employment and was highest in workers exposed to the heaviest concentrations of dust (carders, spinners, weavers, twisters, willowers and pickers). The chief symptoms in workers with asbestosis were progressive dyspnoea, cough, substernal chest pain, blood-streaked sputum, emaciation, weakness and clubbed fingers. Severe dyspnoea, cyanosis and paroxysmal cough were late symptoms. X-ray examination showed characteristically granular or ground-glass markings, especially in the midlung and bases. Emphysema was noted at the apices. Nodular or conglomerate shadows were not observed, but whether this is due to a peculiarity of asbestos dust or to absence of extremely high dust exposure is unknown. Peaking deformities of the diaphragm also occurred less frequently than in silicosis. Asbestosis bodies found in the lungs and sputum appear to be a cellular response to inhaled fibres, and consist of a core of asbestos fibre surrounded by iron-containing protein deposits. The average length is 35 micra. Golden yellow in color, they become brilliant blue when treated with potassium ferrocyanide. They were found on single examination of sputum in 46.9 per cent of workers with asbestosis, but also in 24.3 per cent of workers without clinical or X-ray evidence of asbestosis dust. No cases of asbestosis were found in workers exposed to concentrations below 5 million particles per cu. ft. Modern control equipment is capable of reducing the exposure below this level.—*Asbestosis, R. R. Sayers & W. C. Dreessen, Am. J. Pub. Health, March, 1939, xxix, 205.*—(*H. L. I.*)

**Asbestosis and Tuberculosis.**—The combination of asbestosis and pulmonary tuberculosis is denied by some; by others it is considered frequent, but less frequent than silicosis and tuberculosis. A case in which pulmonary tuberculosis was found associated with asbestosis is described. Asbestos bodies and needles and tubercle bacilli were found in the sputum. Pneumothorax was induced for a cavity in the left lung but was ineffectual because of the extensive fibrosis and rigidity of the lung resulting from the asbestosis. The differential diagnosis of asbestosis from silicosis by X-ray is often difficult. In asbestosis, however, the lesions are generally located in the lower lung fields with a compensatory emphysema in the upper fields, a finding not observed in silicosis, which has been observed most frequently in the middle lung fields. While asbestosis does not influence the course of healed pulmonary tuberculosis, in the presence of active pulmonary tuberculosis it is extremely unfavorable and leads to a more rapidly fatal termination.—*Asbestose und Lungentuberkulose, T. Viegener, Ztschr. f. Tuberk., May, 1939, lxxxii, 295.*—(*A. B. C.*)

**Congenital Cysts of Lung.**—The literature on this subject is comparatively limited as its full recognition comes within the last ten or fifteen years and is due in large part to advances in pulmonary roentgenography although the condition was known as far back as the seventeenth century. The term *congenital cystic disease* implies abnormality of pulmonary tissue with dilatations and cysts not of parasitic or infectious origin. There are two main groups, (*1*) solitary large cysts occurring in early life and often distended, causing pressure symptoms which may lead to asphyxia, and (*2*) the bronchiectatic type, usually multiple and complicated by infection which necessitates appropriate treatment. Cystic disease may be symptomless but chronic respiratory disorders are the rule. The cysts and dilatations are essentially bronchial in character. There is a growing belief that many cases of bronchiectasis have a congenital origin. Sellors here records 32 cases of cystic disease and, collecting cases previously reported, brings the total to well over 400. As many cases must have been overlooked or wrongly diagnosed the condition is probably not rare. Large balloon cysts occur chiefly in the newborn or very young and undergo rapid distension, the lung not only being collapsed but heart and trachea displaced, with respiratory and circulatory embarrassment. The clinical signs are those of tension pneumothorax. Postmortem examination shows the bronchus as a slit-like valve alongside or in the cyst wall, the sac capable only of inflation, not collapse. Roentgenographically there is a clear space

with perhaps some compressed lung tissue or a few fine strands traversing the sac. If it ruptures into the pleura there is a complicating pneumothorax together with the cyst outlines. Solitary cysts are not usually markedly distended. Eventual symptoms are those of suppuration. Associated congenital abnormalities of heart, liver and kidneys may be present. Multiple medium cysts may be symptomless or cause unexplained cough, shortness of breath or small haemoptyses. The individual is apt to be stunted in type, the chest short, deep and asymmetrical, and there may be a faint duskiness or clubbing of fingers. Toward adolescence dyspnoea and asthmatic attacks, recurrent coughs and colds, are common, also haemoptyses. Secondary infection is responsible for the most serious symptoms. The right middle lobe or neighborhood of the right hilum is a favorite location. Roentgenographically there are overlapping rings like the links of a chain and bronchography shows filling of the hollow spaces. Further localization is facilitated by lateral films. Anatomically, the lobar type of multiple small cysts is differentiated from the more disseminated. Clinical symptoms are rarely prominent prior to secondary infection, after which they approximate those of bronchiectasis. The onset is not as clear cut as in the other types and progress often irregular. Complications may dominate the picture, such as pneumonitis, secondary bronchiectasis and abscess formation. Spontaneous pneumothorax may follow rupture of a thin-walled cyst. Cerebral abscess may follow infection. Among conditions to be distinguished are pneumothorax, emphysematous bullae, thin-walled tuberculous cavities and pulmonary abscess, as well as diaphragmatic hernia, hydatid and dermoid cysts. In the toxic type of case caution must be used in employing bronchography and bronchoscopy, preliminary postural drainage being advisable. Drainage of cysts practically never results in closure. Collapse operations are disappointing. Puncture and aspiration of tension cysts afford only temporary relief. Excision of the cystic area is the only effective treatment. Lobectomy and pneumonectomy have resulted in reported cures, but in infected cases preliminary drainage is necessary. They are not warranted in cases with few symptoms. Preliminary bronchoscopy and bronchography are essential. In diffuse cases only palliative measures are possible. There is generally contraction and shrinkage of the diseased areas, varying with the degree of aeration. The essential histological picture is the uniformly bronchial character of the lining membrane. Infection may alter the picture. Communication between bronchi and cysts may be difficult to establish. The stroma is always excessive and irregular. Giant cells, definitely not of a tuberculous character, may be present. Secondary alveolar changes are common. The disruption and derangement of bronchial supporting structures around spaces lined by bronchial type epithelium are almost pathognomonic of a congenital or developmental origin. Clinically and roentgenographically there may be confusion with acquired bronchiectasis. In the present series, the diagnosis of congenital type of lesion has been made only when the dilatations are well beyond the usually recognized dimensions of bronchiectasis and when the X-ray films do not show evidence of long-standing inflammatory change. The most likely pathogenesis is a developmental error of the bronchi. The primitive hilum lies close to the ducts of Cuvier in the lateral wall of the pleuropericardial sac. Another possible factor is atavism or reversion, these changes occurring in an unusually high proportion of cases of accessory or abnormal lobes. The cases reported are accompanied by a considerable number of roentgenographic and bronchographic reproductions and a complete bibliography.—*Congenital Cystic Disease of the Lung, T. H. Sellors, Tubercle, November & December, 1938, xx, 49 & 114.*—(*A. P.*)

**Hydatid Cyst.**—The perforation of a hydatid cyst of the liver into the pleural space and into the lung is very rare. The authors report one such case. The patient, a woman of thirty-five, suffered sudden severe knife-like epigastric pain resembling biliary colic but without nausea or vomiting. Fever and chills followed. The liver was felt four fingers' breadth below the costal margin and was very tender. The spleen was enlarged. There was no jaundice. Several days later signs in the base of the right lung suggested a pleural effusion which was demonstrated by exploratory puncture. A later thoracocentesis revealed a gelatinous amorphous substance without cellular elements and without bacteria. The patient vomited a thick purulent greenish material containing streptococci and pneumococci. Pleurotomy was performed and from the incision exuded a veritable purée of dried withered yellow cysts in some fluid. The patient's sputum resembled the pleural exudate. Lipiodol injected into the pleural space established the topography of the lesion. Surgical intervention was done to close the drainage of the hepatic pocket. A bile stained cystic membrane was removed from a large hepatic cavity. The patient

recovered after an uneventful postoperative course. Dévé in 1935 collected from the literature 40 cases of hydatid cyst rupturing into both the lung and pleura, 82 cases rupturing into the pleura alone and 283 into the lung alone. Dévé distinguishes three anatomical clinical groups: cysts opening into the lung with formation of a pulmonary hydatid cavity and secondary ruptures into the pleural space, cysts opening into the lung and pleura by two distinct orifices, and cysts opening into the pleural space with secondary bronchial evacuation. —*Un cas de kyste hydatique du foie rompu dans la plèvre et le poumon, Constantini, Thiodet & Curtillet, Arch. méd. chir. de l'app. respir., 1938, xiii, no. 5, 392.*—(*J. E. F.*)

**Hydatid Cysts.**—Regarding the radiological signs of hydatid cysts in the lung, Sergent has shown that it is a question of the spherical form or of the distinctness of the contours. Other lesions, such as dermoid cysts, carcinoma, and infarction, may, however, assume an analogous form. Recently, Belot and Peuteuil arrived at an analogous conclusion. These authors feel that the only actual radiological criteria of pulmonary hydatoses are the "sign of separation" in the clinically closed cysts and the "sign of the membrane" in cysts partially evacuated, signs which have been pointed out for some years by foreign physicians. The authors observed 2 cases of hydatid cyst both illustrating the two radiological signs cited.—*A propos de deux aspects radiologiques de pneumo-kystes hydatiques du poumon, P. Pruvost, Grenet & Delore, Arch. méd. chir. de l'app. respir., 1938, xiii, no. 5, 399.*—(*J. E. F.*)

**Hydatid Cysts.**—Surgical intervention for pulmonary hydatid cysts opening into the bronchus, made at an advanced stage is difficult and dangerous (haemorrhages, emboli). The logical treatment of these cysts before emptying is produced is immediate surgery. The hydatid cyst produces an inflammatory reaction in the lung within several hours, appearing on the X-ray film either as fleeting shadows without signs of atelectasis or by opaque shadows persisting for several days, then receding. Following haemoptysis due to the cyst rupturing into the bronchus, transient inflammatory shadows are seen in the lung remote from the location of the cyst. This reaction may be explained on an allergic basis. The cysts are peculiarly surrounded by a dense haemorrhagic zone in the parenchyma. The local haemorrhagic manifestations, analogous to those of haemorrhages in allergy, are dangerous surgically. The hypersensitive state to the hydatid cyst remains for some months even after surgical intervention which explains in part the prolonged pulmonary inflammation. The authors report a case that contributes to the knowledge of the history of hydatid cysts opening into the bronchi. The patient was a thirty-five year old previously healthy male who suffered weakness and somnolence, later followed by a cold and severe bronchopneumonia with fever and an abundant purulent expectoration. The acute illness lasted three weeks, convalesence, three months. A friction rub persisted though clinical recovery followed. A year later an X-ray film of the chest revealed a large cystic shadow in the right upper lobe and a second similar shadow just above the diaphragm. A diagnosis of hydatid cyst was made. The skin test of Casoni was positive. An eosinophilia was present. The Weinberg-Parvu reaction was positive. No other accessible cystic localizations were found. Further observation revealed that only the inferior cyst opened into a bronchus. One day the patient suddenly spat up yellow hydatid liquid. Simultaneously, the patient developed an anaphylactoid reaction, a generalized rash appeared, there was Quincke's oedema of the face and violent dyspnoea set in. Copious amounts of expectoration occurred, mixed with darkly clotted blood and hydatid vesicles. The blood pressure dropped as the patient went into shock. Cardiac stimulants rallied the patient. A marked febrile reaction soon occurred. The barometer was noted to have fallen to 750 mm. at the time the cyst ruptured. The cyst showed a shadow of fluid and air in its interior. There appeared a parenchymatous reaction above the cyst and the interlobar fissure appeared prominent, suggesting an inflammatory reaction. The right upper lobe appeared atelectatic. A haemoptysis occurred. For eight months there was intermittent expectoration of hydatid debris. A course of neosalvarsan was given. The patient remained in fair health for two years when suddenly he began to have haemoptyses of roughly 100 cc.; these persisted daily for two months without a stop. A pleural effusion occurred on the affected side, then gradually disappeared. A haemostatic pneumothorax was started following a large haemoptysis, stopping the bleeding at once. Surgical intervention was instituted. Radiologically, the cystic images were now absent but there persisted fibrous bands. The patient is now clinically well. Casoni's reaction is negative. Had the patient been operated upon before the cysts ruptured into the bronchi, many

dangers could have been avoided.—*Contribution a l'étude des kystes hydatiques pulmonaires ouverts dans les bronches, R. Monod, R. Kourilsky & R. Soberano, Arch. méd. chir. de l'app. respir., 1938, xiii, no. 5, 362.*—(*J. E. F.*)

**Pulmonary Cysts and Emphysematous Bullae.**—Six patients with air-filled pulmonary cavities are reported with the results of investigation from a functional viewpoint. The pulmonary capacity, roentgenographic measurements and analysis of the carbon dioxide and oxygen content of the arterial blood were determined in all cases. The functional changes referable to the respiratory system were similar to those observed in patients with obstructive emphysema. The most common symptoms were dyspnoea and productive cough. The chest volume was larger than normal, the anterior-posterior diameter increased, the expansion greatly diminished and the vital capacity markedly reduced in all but one case. In half of the cases the total capacity was reduced while the residual air was only slightly increased. Evidence is offered which indicates that the volume of the cysts is not included in the residual air in these 4 cases due to poor interchange between tidal air and the gaseous content of the cysts. In the remaining cases the alterations in the pulmonary capacity are typical of obstructive emphysema in which there is a marked decrease in the vital capacity with a corresponding increase in residual air, resulting in a normal value for the total capacity. The ratio of $\frac{\text{residual air}}{\text{total capacity}} \times 100$ is increased in all cases. A slight retention of carbon dioxide was noted in 3 cases, as evidenced by the carbon dioxide content of the arterial blood and some degree of anoxaemia was noted in 4 cases. A reasonably close correlation between the ratio of $\frac{\text{residual air}}{\text{total capacity}} \times 100$ and the oxygen saturation of the arterial blood was found. The tidal volume constituted a much higher percentage of the vital capacity than normal and the pulmonary reserve was reduced. The degree of dyspnoea is closely related to the decrease in vital capacity, the expansion of the chest and the increase in the ratio of $\frac{\text{residual air}}{\text{total capacity}} \times 100$. The dyspnoea can readily be explained by mechanical impairment of the respiratory bellows. The total capacity and residual air are increased when a patient assumes a sitting position and the carbon dioxide percentage of expired and alveolar air is increased. The question arises in this group, as in others, whether the condition is congenital or acquired. After reviewing the cases the authors conclude that it is hard to determine the part played by chronic pulmonary infection and that, from a clinical standpoint, it is often impossible to distinguish between congenital cystic disease of the lung and emphysematous bullae. They also feel that roentgenographic studies do not always parallel the respiratory function so that functional tests are frequently indicated to determine the degree of disability.—*Pathological Physiology of Pulmonary Cysts and Emphysematous Bullae, N. L. Kaltreider & W. W. Fray, Am. J. M. Sc., January, 1939, cxcvii, 62.*—(*G. F. M.*)

**Cyst of Lung.**—In a baby of two months, which had had repeated attacks of screaming, struggling for breath and cyanosis, classical signs of right-sided pneumothorax were found, but careful X-ray study led to a diagnosis of inflated lung cyst, probably in the middle lobe. Deflation of the cyst by trocar and cannula during a critical congestive attack, though followed by immediate relief, led to pneumothorax by leakage of air through the puncture wound in the cyst. Retraction of the cyst together with coughing led to slipping out of the cannula, and then the cyst refilled with air. Life being again threatened, the cyst was again deflated. The chest was then opened in the fourth interspace, disclosing the cyst looking like a purple balloon. The opened cyst was sewn to the incision and a drainage tube inserted. Unfortunately the parts became infected, empyema and bronchopneumonia ensued, and the infant soon died. A columnar cell lining was found in sections of the cyst wall. The lung was so much damaged on removal, owing to the numerous adhesions, that the exact connections of the cyst could not be determined. The case illustrates the dangers of mistaken diagnosis and treatment. Differentiation from pneumothorax is enjoined. Fortunately such cases are rare. If untreated, death may occur in a congestive attack. The ideal treatment is total removal of the cyst and even pneumonectomy.—*Single Large Cyst of the Lung Simulating a High-Pressure Pneumothorax, E. H. Hudson, Brit. M. J., March 11, 1939, 503.*—(*C. C. M.*)

**Mediastinal Emphysema.**—A series of 7 unusual cases are described all having in common curious crackling, crunching sounds in the region of the heart following an acute painful onset. These sounds conveyed the impression of air being churned or squeezed about in the tissues. The sounds would usually vanish with a change in posture.

In some instances there was a swelling above the clavicle with subcutaneous crepitations. In none of the cases was there any evidence of shock. Constitutional symptoms were at a minimum. All made a rapid recovery. In these cases the author concludes that the unusual symptoms described are due to the escape of air into the interstitial tissue of the lungs, the air quickly reaching the mediastinum. Apparently the pulmonary alveoli of healthy persons may rupture from unknown causes and allow air to escape. The escaped air usually travels along the fascial planes toward the mediastinum, although it may go toward the visceral pleura. After having reached the hilum the air spreads out in the mediastinum, infiltrating the tissues that lie between the heart and the mediastinum. The air may be trapped in the mediastinum and be absorbed, or it may burrow upward and appear above the clavicles, spreading up the neck and over the front of the chest. In 2 out of the 7 cases reported a pneumothorax was present. There is much evidence to suggest that the pneumothorax is produced by escape of air from the mediastinum, but the possibility of the rupture of a pleural bleb cannot be ignored. The spontaneous occurrence of interstial emphysema of the lung and mediastinal emphysema has not heretofore been recognized and the curious resulting symptoms have never been clearly described or their significance emphasized. The following is a summary of the essential clinical features and important symptoms of spontaneous interstitial emphysema of the lungs and mediastinum: (*1*) Interstitial emphysema of the lung may occur without the least effort, when the patient is quietly standing, sitting or lying down. (*2*) When the air reaches the mediastinum, distending the mediastinal tissues, the patient complains of pain which is often very severe. Usually the pain is located beneath the sternum; sometimes it radiates to the back; at other times to the neck and shoulders, rarely to the arms. Accompanying the pain there is often a sensation of pressure or of expansion beneath the sternum. (*3*) There are no constitutional symptoms, no evidence of shock. The temperature, the pulse and respiratory rates, the blood pressure, the leucocyte count are very little altered if any. (*4*) In many instances a peculiar and distinctive sound is heard over the heart synchronous with its contractions. Usually the sound is heard only during systole but at times it may be heard also during diastole. (*5*) The area of cardiac dulness is diminished or completely obliterated, the dulness being replaced by a hyperresonant percussion note. (*6*) Pneumothorax often occurs. The pneumothorax is usually small and may not be suspected until a roentgenogram of the chest has been taken. Pneumothorax was discovered in 2 of the 7 cases observed, in both instances on the left side. (The author sees no reason, other than accidental, why it should have been on the left side and is inclined to predict that in other cases it will be found upon the right.) (*7*) The roentgenogram is a valuable aid in establishing the diagnosis. In instances in which the characteristic sound over the heart is absent the roentgenographic evidence of air in the mediastinum may be decisive. The most convincing proof of this is seen in the lateral roentgenograms when the tissues between the heart and anterior wall of the chest are infiltrated with air. If such proof cannot be obtained the anteroposterior film must be carefully inspected for evidence of air in the mediastinum. The evidence consists of a sharp and distinct line running parallel to the border of the heart. (*8*) When air appears in the subcutaneous tissues of the neck the diagnosis is at once assured.—*Spontaneous Mediastinal Emphysema, L. Hamman, Bull. Johns Hopkins Hosp., January, 1939, lxiv, 1.*—(*J. S. W.*)

**Interstitial Emphysema.**—A syndrome resulting from the spontaneous development of interstitial emphysema of the lungs, first fully described by Louis Hamman in 1937, is characterized by the sudden development of severe pain in the chest without any preceding trauma or unusual exertion and a "peculiar crunching, crackling bubbling sound heard over the heart with each contraction." There may also be a decrease or disappearance of the area of cardiac dulness, air visible by X-ray in the mediastinum or pneumothorax, especially on the left. Subcutaneous emphysema, when present, makes the diagnosis obvious. In some cases the characteristic sounds may be heard several feet from the patient. They vary with change in position and show great variation in quality and intensity during the course of the illness. The patient may call the physican's attention to the peculiar noises in his chest. Movement, especially sudden movement as in sneezing or coughing, may be quite painful. This condition is due to rupture of alveoli or bronchioles permitting the escape of air into the interstitial tissue of the lungs. If the amount of air is large it may dissect along the interstitial bands to the hilum and enter the mediastinum or pass outward to the pleura forming a bleb which may rupture, producing pneumothorax. In some instances the air travels in both directions. The characteristic sounds are probably due to the im-

pingement of the contracting heart on the air collected in the areolar tissue of the anterior mediastinum and also to subpleural blebs in the region of the heart. Only 2 certain cases have been reported since 6 were described by Hamman. Therefore, a report is made of another similar case and also one which occurred in a patient with rheumatic heart disease following labor. The picture seems to be identical whether the condition is spontaneous or secondary. It is felt that spontaneous pulmonary emphysema is not rare and that if all cases of spontaneous pneumothorax were examined carefully the typical sounds would often be found. It may also be produced artificially by puncture of the lung.—*Spontaneous Interstitial Emphysema of the Lungs, J. McGuire & W. B. Bean, Am. J. M. Sc., April, 1939, cxcvii, 503.*—(*G. F. M.*)

**Mediastinal Emphysema.**—Attention was first called to this rare condition by Hamman who, in 1937, reported 5 cases. In the same year Scott reported 2 cases and noted the similarity of the pain to angina or coronary thrombosis. Few cases are reported in the medical literature. Mediastinal emphysema sometimes occurs alone or with pneumothorax as the result of a definite cause. It may occur following influenza, pneumonia, trauma to the trachea or pleura, thoracotomy and thyroidectomy or artificial pneumothorax when air is injected directly into the lung tissue. Severe asthma may precipitate it. Strains of any kind such as lifting, crying or coughing may result in rupture of the alveolus. Though this would seem to be a rare condition, it may occur more often than one would think and be responsible for some chest pains occurring without apparent cause. A correct diagnosis is of importance because of the seriousness of the conditions it may simulate. It must be differentiated from coronary thrombosis, acute pericarditis, ruptured aneurysm and pulmonary embolus. Following rupture of an alveolus, air dissects its way through the fibrous tissue about the bronchi and blood vessels, escaping into the mediastinum through the hilum of the lung. If sufficient air accumulates it may dissect its way into the neck and over the shoulders. Occasionally it may dissect its way to the pleural cavity producing spontaneous pneumothorax. The onset is usually sudden. Pain is severe, is present along the border of the sternum and is referred to the shoulder and arm. The pulse is increased and occasionally the temperature is elevated. Dyspnoea or cyanosis may be present. Soon after onset of the pain the patient frequently can hear a clicking or knocking in the chest, which may be associated with respiration, heart beat or even with swallowing. The intensity of the noise is altered by changes in position. Physical examination reveals the patient to be in pain but not in collapse. He may lean forward being most comfortable in that position. Emphysema may be felt in the neck or shoulders. The area of cardiac dulness is diminished or absent, but the most characteristic finding is the noise heard in the chest which resembles a knock, click or squeak of a leather saddle and may be heard some distance from the patient without the aid of the stethoscope. It may be synchronous with the heart beat, respiration or both and will vary with change in position. The X-ray film may show an area of pneumothorax along the lateral borders of the mediastinum and heart. No treatment other than rest and sedatives is required, with graded exercise in two or three weeks. In more serious cases in which air hunger and cyanosis are present due to pressure on the mediastinal blood vessels venesection and artificial pneumothorax have been suggested. A typical case of mediastinal emphysema is reported. The patient, a medical student, was seized with severe pain while working in the laboratory. The pain was so severe he could not continue working. The following morning the pain was more severe, and a clicking sound was audible a foot from the patient. The roentgenogram revealed a small area of pneumothorax on the left with most of the collapse at the apex. A small amount of free air was present along the left cardiac border. Convalescence was uneventful. A similar episode, but not so severe, occurred several weeks later following exercise.—*Spontaneous Mediastinal Emphysema, J. B. Morey & M. C. Sosman, Radiology, January, 1939, xxxii, 19.*—(*G. F. M.*)

**Spontaneous Haemopneumothorax.**—The occurrence of spontaneous haemopneumothorax on the contralateral side of a patient who has had a thoracoplasty is very rare. The patient whose history is reported had bilateral pulmonary tuberculosis for which pneumothorax had been induced on the right. A left thoracoplasty was then done and six days after operation she had a massive spontaneous pneumothorax on the right. Four weeks later a second-stage thoracoplasty was performed and seven months later she was in excellent condition. One day after a refill of the right pneumothorax she again had a right spontaneous haemopneumothorax; the large blood clot slowly resorbed. The aetiology is probably similar to that of spontaneous pneumothorax without haemorrhage.—*Report of a Case of a*

*Thoracoplasty with a Spontaneous Hemopneumothorax on the Contralateral Side, A. A. Weiner & A. Jackson, Quart. Bull. Sea View Hosp., January, 1939, iv, 211.—(A. A. E.)*

**Spontaneous Pneumothorax.**—In late years it is being recognized that pneumothorax occurs in apparently healthy persons. The true incidence of idiopathic spontaneous pneumothorax is difficult to determine because the condition frequently occurs with little or no symptoms. In the author's study, 15 cases of pneumothorax proved by roentgenograms have been recognized from symptoms in an average male student body of 2,500 at the University of Virginia. Eleven of these cases have occurred in five years. Exertion *per se* is not a necessary precipitating factor. In at least 3 instances the first symptom was noted while the patient was relatively quiet. When symptoms occur, their onset is usually very abrupt. Pain is generally the first symptom. It may require morphine, or be so mild that it is described as "oppression." Although the pain persists between respirations, it is ordinarily exaggerated by a deep inspiration. The intensity may increase for several hours before it gradually subsides. Dyspnoea may be absent, mild or marked. It may occur as an early symptom or be noted only several days later. Cases presenting a febrile course are presumptively not due to idiopathic pneumothorax. Careful chest films furnish the most exact method of diagnosis. Fluoroscopic examination alone is not reliable. No acid-fast organisms were found in this series. In college students the chief problem in differential diagnosis is to exclude clinical tuberculosis. No physical or X-ray signs suggestive of tuberculous activity were found in these cases, although several films exhibited calcium deposits. There have been 3 cases of recurrent pneumothorax. One patient had five episodes of pneumothorax on the same side during his college career but has had no recurrence in thirteen years. Rest in bed is all the treatment usually required. The period depends somewhat on the degree of pneumothorax, but since the author's experience with the condition has increased the period of bed-rest has shortened. Tension pneumothorax requires immediate puncture, which may have to be repeated. Aspiration may be tried in chronic pneumothorax. The prognosis is excellent. The condition may recur once or several times, but the patient survives. Although tuberculosis may be a common cause of the scar tissue to form the valve-like mechanism necessary to the vesicle formation in pneumothorax, it is extremely rare that clinical tuberculosis develops as a sequela. Fourteen of the author's 15 patients have been followed and are known to be in good health after periods ranging from eight months to fifteen years.—*Spontaneous Pneumothorax in College Students, S. D. Blackford, J. A. M. A., August 26, 1939, cxiii, 737.—(G. L. L.)*

**Disk-like Atelectasis.**—In various abdominal conditions linear shadows, usually straight and clearly defined but occasionally slightly triangular in shape, may be seen crossing the bases more or less horizontally, resembling thickened interlobar septa except for their location. They may be single or multiple and occur on one or both sides of the chest. The corresponding hemidiaphragm is usually elevated and a small pleural effusion may be present. These shadows persist for indefinite periods. When seen, they are suggestive of abdominal disease. In the absence of autoptic findings it was assumed that such shadow lines were circumscribed pleural thickenings, but recently Fleischner described their true character, calling them plate-shaped or disk-like atelectases. They apparently occur when respiration becomes shallow, especially in the presence of increased bronchial secretion. Their linear appearance is due to the fact that a small area of atelectasis tending to retract, but surrounded by normal lung, is pulled out by the negative intrapleural pressure and relative rigidity of the thoracic wall. Mobile organs may be displaced by such areas of atelectasis. In most cases the condition is transient for it either resolves or is followed by pneumonic infiltration. Because of its transient nature and previous lack of attention to this condition, data concerning the incidence are not available. The author found 6 cases in 1,000 examinations made in 1936 and 11 cases in 482 more recent examinations. In his paper, Fleischner reported 2 cases confirmed by autopsy. The author reports a third and gives the clinical and roentgenographic findings in illustrative cases.—*Disk-like Atelectases, A. Oppenheimer, Radiology, December, 1938, xxxi, 651.—(G. F. M.)*

**Atelectasis and Pneumothorax.**—The greatest danger in pulmonary atelectasis is the secondary development of pneumonia. It is, therefore, important either to prevent atelectasis or to relieve it as soon as possible. Present methods of therapy aim at releasing the secretions that may be obstructing the bronchi of the affected lung. Very few cases have been reported in the literature of the relief of massive atelectasis by artificial pneumothorax. The authors report the case of a male Negro patient with tuberculosis

who suddenly developed a massive atelectasis of the left lung with acute symptoms, becoming progressively worse within twelve hours, at which time 500 cc. of air was injected into the pleural cavity. Within twenty-four hours dyspnoea and cyanosis had disappeared and there was no recurrence. Since all patients with pulmonary atelectasis show increased negative intrapleural pressure, it seems likely that the cyanosis and dyspnoea are due primarily to a displacement of the heart and great vessels and to an acute overdistention of the unaffected lung. In cases of atelectasis following abdominal operations, the marked increase of negative pressure draws the entire obstructing medium more firmly and deeply into the bronchus, thus perpetuating a vicious cycle. Introducing air into the pleural cavity serves to break this cycle, tends to push the mediastinum back to its original position and enhances the expulsion of the obstructing medium.—*Massive Pulmonary Collapse Treated with Artificial Pneumothorax; Report of a Case, H. G. Jacobson, S. Gendelman & D. Goldman, J. A. M. A., June 3, 1939, cxii, 2249.*—(*G. L. L.*)

**Lipoid Pneumonia.**—Lipoid pneumonia, now recognized as a disease entity, is not uncommon. A discussion is, therefore, presented based upon 27 cases observed at the Children's Hospital of Philadelphia. The disease often goes unrecognized because no roentgen findings pathognomonic of the condition have been noted. In but one of 20 children autopsied was the true nature of the pulmonary lesion determined, though 16 had had X-ray films of the chest. In 7 patients the diagnosis was based upon history, physical examination and roentgen findings. Infants with some kind of constitutional debility and neurological disorder seem to be especially susceptible. All but 4 of the children studied were under two years of age, most were under one year of age and in two-thirds of the cases there was an associated constitutional disturbance. The retardation of growth and poor nutrition observed in 21 of these patients were attributable to such a disturbance rather than to the pneumonia. None had a positive tuberculin test. The location of the lung lesions seen at necropsy clearly showed the exogenous source of the lipoid. The posterior dependent portions of the lung and perihilar areas were always affected and the right lung was more involved than the left. Occasionally the left lung was unaffected. The aspirated oil apparently was codliver oil though petrolatum from nose drops may have been the causative agent in several cases. Ikeda felt that a diagnosis might be attempted with the help of an accurate history and roentgenograms of the chest. In the cases reviewed a marked variation in X-ray findings was noted. Roentgenological differentiation of pathological stages is impossible, but the lesions could be classified as mild, moderate and severe. The extent of involvement depends upon the amount of oil in the lung and no differentiation as to the type of oil aspirated could be made from roentgen studies. Mild involvement is seen as an increase in the density and extent of the hilar shadows and there may be scattered areas of increased density along the descending right bronchus branches. When the involvement is moderate the perihilar shadows are widened in all directions. In severe cases the perihilar shadows are spread out well toward the periphery of the lung and may extend to the pleura, especially posteriorly. However, there usually is a clear zone between the perihilar shadow and the chest wall which increases in width as the child grows older. Serial films are needed to demonstrate the development and chronicity of the disease. A definite diagnosis cannot be made in mild or moderately severe cases without a history of oil aspiration. In the reaching of a diagnosis bronchosinusitis, tuberculosis, rachitic atelectasis and the increased markings of congenital cardiac disease would have to be considered. It is obviously injudicious to use oil as nose drops in small or weak children or to force codliver oil or liquid petrolatum.—*Lipoid Pneumonia in Infants and Children, R. S. Bromer & I. J. Wolman, Radiology, January, 1939, xxxii, 1.*—(*G. F. M.*)

**Pulmonary Artery Arising from Abdominal Aorta.**—With the development of thoracic surgery accessory pulmonary arteries acquire greater significance. Accessory pulmonary arteries arising from the thoracic aorta have been reported in the literature some thirteen times, from the abdominal aorta once, from an intercostal artery once, and from the right phrenic artery once. In the present case an artery 8 mm. in diameter and 9 cm. in length was found to have its origin in the upper part of the coeliac axis. In addition to the pulmonary branch, the coeliac axis gave off right and left phrenic arteries and the usual left gastric, hepatic and lienal arteries. The pulmonary branch appeared to pierce the diaphragm through a hiatus of its own on a level with the oesophageal opening and about 1 cm. medial to it. It was not accompanied by other structures. The artery supplied the posteroinferior portion of the lower lobe of the right lung.—*A Pulmonary*

*Artery Arising from the Abdominal Aorta, M. Batts, Jr., J. Thoracic Surg., June, 1939, viii, 565.—(L. F. B.)*

**Hodgkin's Disease.**—An enlargement of mediastinal lymph nodes producing pressure signs with eventual involvement of the lung is usually brought to mind when intrathoracic Hodgkin's disease is mentioned. Partial or complete atelectasis would be anticipated as the result of pressure of the enlarged lymph nodes. However, on reviewing the literature, partial atelectasis was found to occur infrequently and reports of only 2 cases of massive atelectasis were discovered. In these 2 cases autopsy revealed that the obstruction was not due to pressure but to actual invasion of the bronchial walls. The author presents a case of Hodgkin's disease complicated by massive collapse, pleural effusion and cavitation. The first roentgenogram showed extensive infiltrations in the lower two-thirds of the right lung, multiple areas of bronchiectasis and an area at the level of the eighth rib appearing to be an area of necrosing pneumonitis, a thickened pleura near the right hilum and partial atelectasis of the right lung. At bronchoscopy an inflammation of the right main bronchus and thick pus characteristic of bronchiectasis were found. Temporary clearing of some of the disease on the right occurred following the first course of X-ray over the lung. A second course exaggerated the symptoms and a roentgenogram shortly after revealed a massive collapse of the right lung so that no more X-ray therapy of the lung was given. Eight months later two cavities were seen in the right apex and there was beginning infiltration at the left hilum. The degree of atelectasis varied greatly from time to time, clearing in the upper lung field only to reappear in the lower lobe of the lung and vice versa. The postmortem examination revealed marked enlargement of the mediastinal and bronchial lymph nodes, obliteration of the right pleural cavity, a large cavity filled with green pus occupying most of the right upper and middle lobes, complete atelectasis of the right lower lobe, extensive infiltrations and numerous cavities in the left lower lobe and narrowing of the right main bronchus to about 3 mm. in diameter. The obstruction apparently was due to the tenacious mucopurulent sputum arising from the severe bronchitis secondary to direct involvement of the bronchial tubes by Hodgkin's disease. Thus the partial constriction of the bronchus was changed at various times to complete obstruction by the thick sputum. *Monilia albicans* was present in a culture of the sputum, but no pathological significance is attached to this finding.—*A Case of Hodgkin's Disease with Massive Collapse and Cavitation of the Lung, B. L. Hardin, Am. J. M. Sc., January, 1939, cxcvii, 92.—(G. F. M.)*

**Intrathoracic Thyroid.**—Clinically, thyroids fall into two main groups: one in which the thyroid assumes a position behind the sternum and the other in which there are aberrant glands either attached to the main gland by fibrous tissue or completely separated thyroid rests within the thyroid cavity. Twelve to 30 per cent of goitres are found to be retrosternal, and approximately 80 per cent of these occur in women between fifteen and thirty-five years of age. Large intrathoracic goitres may remain symptomless or, on the other hand, may give signs and symptoms of mediastinal pressure. The roentgenological differential diagnosis includes sacculated aneurysm of the ascending aorta, persistent thymus, lymphosarcoma, Hodgkin's disease, other mediastinal tumors and echinococcus cyst. An unusual case is presented. A forty-nine year old man had complete exhaustion followed by a sense of pressure in the throat and right side of the neck, violent coughing attacks productive of blood, dyspnoea, palpitation, moderate dysphagia and physical signs of a tumor in the right thoracic space. Three and one-half years later he appeared desperately ill and presented evidence of recent weight loss, dyspnoea, cyanosis, enlarged veins over the upper abdomen and sternum, a palpable mass in the right supraclavicular region and physical signs of lesions in the right side of the chest. Roentgenography showed a tumor composed of four distinct masses, each surrounded by a thin wall of calcium: the upper mass was centrally and anteriorly located and extended about 4 cm. into both lung fields; a smaller mass was in the upper half of the left midlung field, a large mass occupied about two-thirds of the right lung field and a fourth small mass (visualized by Potter-Bucky diaphragm) was situated just above the base of the heart. A definite preoperative diagnosis was not made. The mass in the right supraclavicular region proved to be the superior pole of a substernal thyroid. The right lobe was removed and found to be nonmalignant. The rest of the thyroid was removed with some difficulty since it was firmly attached to the chest wall and all surrounding structures. The patient could not tolerate the tremendous change in intrathoracic pressure.—*Large Intrathoracic Thyroid, Case Report, R. B. Miller, Am. J. Roentgenol., July, 1938, xl, 66.—(E. M. J.)*

**Treatment of Addison's Disease by Implantation of Hormone.**—In previous experiments it was noted that bilaterally adrenalectomized dogs could be maintained in excellent condition by implanting, subcutaneously, pellets of synthetic adrenal cortical hormone (crystalline desoxy-corticosterone acetate). The pellets were slowly absorbed, hence their action was prolonged. Removal and weighing of pellets indicated the rate of absorption which did not vary considerably in different animals. In these studies the number and size of pellets necessary for maintenance was compared with the daily requirements of the artificial hormone in oil injected subcutaneously. The implantation of the drug was found to result in a considerable saving in hormone (20 to 30 per cent). Furthermore, the number of pellets required for maintenance could be predicted rather accurately from the daily requirement of hormone in oil. In the light of this knowledge 6 patients with Addison's disease were selected for treatment with pellet implants. None of these patients could be maintained in good condition by means of sodium therapy alone. All showed pigmentation. Only one gave definite evidence of tuberculosis. This patient showed tubercle bacilli in his urine as well as roentgen ray evidence of the presence of this disease. Since the supply of crystalline synthetic hormone available for this study was limited, added sodium chloride therapy was given to 5 of the 6 patients as it has been shown that added sodium chloride (8 to 12 g. daily) greatly reduces the requirement of the hormone. The 6 patients were provided with a diet of constant mineral intake. The daily maintenance dose of the hormone in sesame oil, injected once daily, was determined. The maintenance of optimum body weight, normal blood pressure, normal blood volume, positive sodium and chloride balance and normal concentration of plasma electrolytes was considered as evidence of adequate treatment. The daily injections of hormone in oil were then discontinued, and, under local anaesthesia, pellets of the synthetic hormone weighing 50 to 150 mg. each were inserted subcutaneously in the infrascapular region. The pellets were prepared by compressing the sterile crystalline hormone in a suitable mold. No binding substance was employed. The pellets measured 6.5 mm. in diameter by 2 to 6 mm. in thickness. Silk thread or silver wire was attached to the pellets to facilitate localization at a later date. The decrease in the weight of the pellets measured the amount of the hormone absorbed. Careful observations indicated that one pellet of 100 to 150 mg. is required for each 0.5 mg. of hormone in oil necessary for maintenance. The pellets themselves caused no local discomfort or reaction. Pellets weighing 50 to 150 mg. are absorbed at the rate of 0.25 to 0.35 mg. per day and this rate was found to be quite constant even as the pellet decreases in size. This uniformity in absorption is probably accounted for by either a softening of the pellet which would result in more rapid absorption, or canalization which would offer a greater surface for absorption. Thus it is possible by the insertion of the required number of pellets to provide adequate hormone therapy for at least six to ten months. The hormone requirement of a patient does not appear to modify the rate of absorption. The number of pellets inserted at the beginning of treatment ranged from two to six per patient. Three cases later received extra pellets. The implantation of the pellets as described, besides providing a daily maintenance dose of the hormone, resulted in a striking and continued improvement in all the patients. Some decrease in pigmentation occurred in three. This method of treatment obviates the necessity for daily injections of the hormone, and, in addition, effects a great economy in its use. It appears to be the most efficacious manner available for administering the synthetic hormone. Pellet implants alone are not a suitable form of treatment for patients in crisis. It may be necessary to supplement pellet therapy with injections of the hormone, an increase in the intake of sodium chloride and a reduction in potassium intake during the course of an acute infection. Complete case protocols accompany the article.—*Treatment of Addison's Disease with Pellets of Crystalline Adrenal Cortical Hormone (Synthetic Desoxy-corticosterone acetate) Implanted Subcutaneously, G. W. Thorn, R. P. Howard, K. Emerson, Jr. & W. M. Firor, Bull. Johns Hopkins Hosp., May, 1939, lxiv, 339.—(J. S. W.)*

**Nontuberculous Erythema Nodosum.**—During the first half of 1937 a rather extensive, close succession of cases of erythema nodosum in which the tuberculin reaction was negative occurred in Stockholm. During the first six months of that year 34 patients with erythema nodosum were received at the children's hospitals, 20 of whom had negative tuberculin reactions. In these cases the condition occurred during the months of April and May, while in the cases in which the tuberculin reaction was positive the incidence was evenly distributed over the period of January to June. In comparison, during the years 1928 to 1936, only 34 of 420 patients treated for erythema

nodosum had negative reactions to tuberculin. The 20 cases of nontuberculous erythema nodosum were subjected to a close analysis, which gave the following results. Heredity and exposure to tuberculosis played an exceedingly small part. Mantoux tests from 1 to 20 mg. of tuberculin were carried out with negative results. Roentgen examination of the lungs was made in all these cases, and no changes, or merely unimportant ones were proved; at any rate they were not of the scope generally associated with tuberculous erythema nodosum. In one case the condition was complicated with pneumonia and empyema and the course ended with death. This case is of special interest, for as far as is known it is the first case of erythema nodosum associated with a negative tuberculin reaction in which postmortem examination was made. At the autopsy, which was carried out with special consideration for the possible existence of tuberculosis, no tuberculous changes could be traced.—*Close Succession of Cases of Erythema Nodosum of Nontuberculous Pathogenesis, H. Ernberg & O. Gabinus, Am. J. Dis. Child., May, 1939, lvii, 1012.*—(*From the Authors' Summary*)—(*M. B.*)

**Treatment of Lupus Erythematodes with Chrisalbine.**—In contradistinction to lupus vulgaris, lupus erythematodes does not respond to sunlight, X-ray or radium treatment. Ten patients have been treated with chrisalbine, a thiosulfate compound of gold and sodium bicarbonate. In all patients a more or less rapid and complete regression of the foci was noted. The usual precautions of any gold treatment are necessary. Regular examination and control of the nondiseased skin areas seem advisable. —*Die Goldbehandlung des Lupus erythematodes mit Chrysalbine, W. Friedländer, Schweiz. med. Wchnschr., June 3, 1939, xxii, 500.*—(*A. B. T.*)

**Inguinal Granuloma.**—A woman of fifty-two who at the age of twenty had a tuberculous "apicitis" developed an inguinal lymph node of about the size of a fist which began to grow within one year before examination. A similar swelling on the opposite side had been removed previously. The deep lymph nodes of the iliac region were hard, slightly swollen, and tender. The blood picture showed 15 per cent monocytes. Pirquet and Frei tests were positive. After inoculation of sterile pus into the peritoneal cavity of guinea pigs a generalized tuberculosis developed, and following injection of pus into the groin of guinea pigs large lymphatic swellings occurred which histologically showed granulation tissue. The authors believe that the inguinal lymphadenitis was due both to tuberculosis and to lymphogranuloma. The generalized adenopathy and a previous tuberculous lesion of the patient make a tuberculous infection highly probable although neither clinical nor histological signs have proved it definitely.—*Strumöser Bubo tuberkulösen und lymphogranulomatösen Ursprungs, N. Melczer & K. Sipes, Acta dermat.-venereol., March, 1939, xx, 135.*—(*F. G. K.*)

**Key to Abstractors**

*A. A. E.:* Adrian A. Ehler, Albany, New York.
*A. B. C.:* Adele B. Cohn, Bedford Hills, New York.
*A. B. T.:* Alice B. Tobler, Bedford Hills, New York.
*A. P.:* Andrew Peters, Springfield, Massachusetts.
*C. C. M.:* C. C. Macklin, London, Canada.
*E. M. J.:* Edna M. Jones, Northville, Michigan.
*F. G. K.:* F. G. Kautz, New York City.
*G. F. M.:* Gertrude F. Mitchell, Detroit, Michigan.
*G. L. L.:* George L. Leslie, Howell, Michigan.
*H. L. I.:* Harold L. Israel, Philadelphia, Pennsylvania.
*H. R. N.:* H. R. Nayer, New York City.
*J. E. F.:* Jason E. Farber, Buffalo, New York.
*J. S. W.:* J. Stanley Woolley, Liberty, New York.
*L. F. B.:* Lauren F. Busby, Northville, Michigan.
*M. B.:* Miriam Brailey, Baltimore, Maryland.

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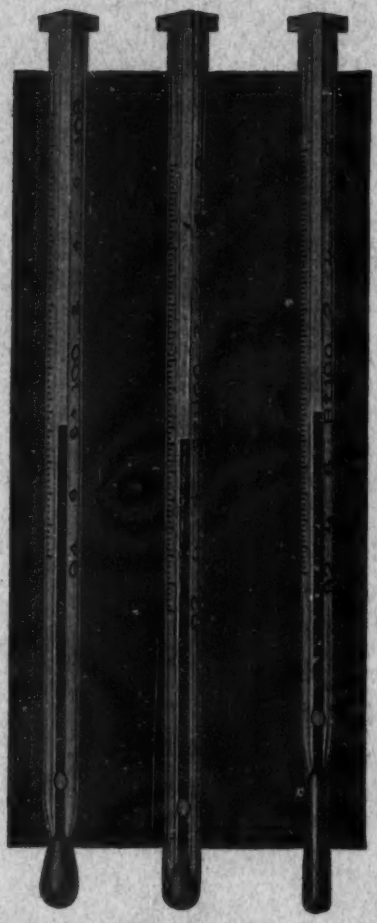

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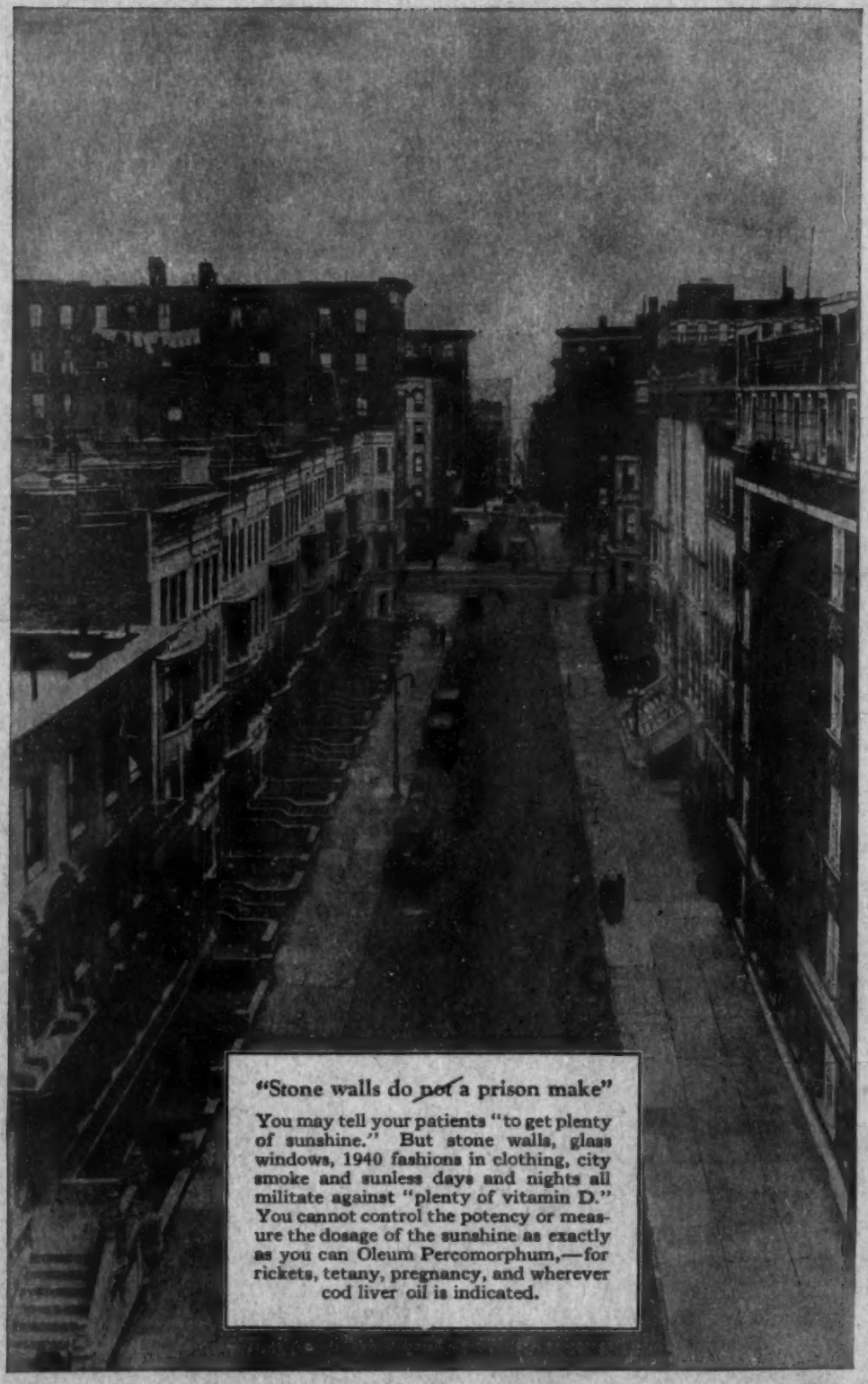

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